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6: Matusick-Kumar L, Hurlburt W, Weinheimer SP, Newcomb WW, Brown JC, Gao M. Related Articles, Links

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7: DiIanni CL, Stevens JT, Bolgar M, O'Boyle DR 2nd, Weinheimer SP, Colonno RJ. Related Articles, Links

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Arch Virol. 2000;145(12):2677-86.
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The equine herpes virus 4 thymidine kinase is a better suicide gene than the human herpes virus 1 thymidine kinase.
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5: Lewis JB, Thompson YG, Feng X, Holden VR, O'Callaghan D, Caughman GB. Related Articles, Links

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Virology. 1997 Apr 14;230(2):369-75.
PMID: 9143293 [PubMed - indexed for MEDLINE]

6: Feng X, Thompson YG, Lewis JB, Caughman GB. Related Articles, Links

Expression and function of the equine herpesvirus 1 virion-associated host shutoff homolog.
J Virol. 1996 Dec;70(12):8710-8.
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```
=> "herpes virus"
         20676 "HERPES"
        273877 "VIRUS"
         52420 "VIRUSES"
        283178 "VIRUS"
                 ("VIRUS" OR "VIRUSES")
L1        6338 "HERPES VIRUS"
                 ("HERPES"(W)"VIRUS")

=> " VP16 deletion" and L1
          1573 "VP16"
         64214 "DELETION"
         21169 "DELETIONS"
         74621 "DELETION"
                 ("DELETION" OR "DELETIONS")
             2 " VP16 DELETION"
                 ("VP16"(W)"DELETION")
L2           0 " VP16 DELETION" AND L1
```

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=> Vmw65 adj deletion
           347 VMW65
           202 ADJ
         64214 DELETION
         21169 DELETIONS
         74621 DELETION
                 (DELETION OR DELETIONS)
L3           0 VMW65 ADJ DELETION
                 (VMW65(W)ADJ(W)DELETION)

=> Vmw65 (w) deletion
           347 VMW65
         64214 DELETION
         21169 DELETIONS
         74621 DELETION
                 (DELETION OR DELETIONS)
L4           0 VMW65 (W) DELETION

=> Vmw65
L5         347 VMW65

=> L1 and L5
L6          76 L1 AND L5

=> "10 protein"
       3234115 "10"
       1446895 "PROTEIN"
        957194 "PROTEINS"
       1670799 "PROTEIN"
                 ("PROTEIN" OR "PROTEINS")
L7        1759 "10 PROTEIN"
                 ("10"(W)"PROTEIN")

=> L7 and L1
L8           4 L7 AND L1

=> transactivation and L6
          8211 TRANSACTIVATION
            17 TRANSACTIVATIONS
          8218 TRANSACTIVATION
                 (TRANSACTIVATION OR TRANSACTIVATIONS)
L9           16 TRANSACTIVATION AND L6

=> transactivation and L8
          8211 TRANSACTIVATION
            17 TRANSACTIVATIONS
          8218 TRANSACTIVATION
                 (TRANSACTIVATION OR TRANSACTIVATIONS)
L10          0 TRANSACTIVATION AND L8

=> ICP4 and L7
           499 ICP4
L11          3 ICP4 AND L7

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L11 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:239782 CAPLUS
DOCUMENT NUMBER: 120:239782
TITLE: Varicella-zoster virus (VZV) virion-associated transactivator open reading frame 62 protein enhances the infectivity of VZV DNA
AUTHOR(S): Moriuchi, Masako; Moriuchi, Hiroyuki; Straus, Stephen,
E.; Cohen, Jeffrey I.
CORPORATE SOURCE: Lab. Clin. Invest., Natl. Inst. Allergy Infect. Dis., Bethesda, MD, 20892, USA
SOURCE: Virology (1994), 200(1), 297-300
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Varicella-zoster virus (VZV) open reading frame (ORF) 62 protein (the homolog of herpes simplex virus type 1 (HSV-1) **ICP4**) and ORF **10 protein** (the homolog of HSV-1 VP16) are virion-assocd. transactivators. To investigate whether these proteins function during the initial stages of VZV infection, human melanoma cells were cotransfected with purified VZV DNA, devoid of any structural proteins, along with a plasmid expressing VZV ORF62 or ORF10 under the control of the human cytomegalovirus major immediate-early promoter. Expression of ORF62 enhanced the infectivity of VZV DNA up to 70-fold. In contrast, expression of ORF 10 enhanced the infectivity of VZV DNA only threefold. These results show that high-level expression of ORF62 protein increases the probability that transfected VZV DNA will result in productive infection, suggesting that this virion-assocd. transactivator (ORF62) has a crit. role in initiating infection.

L11 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:45928 CAPLUS
DOCUMENT NUMBER: 120:45928
TITLE: Methods and compositions for gene, tumor, and viral infection therapy and prevention of programmed cell death (apoptosis)
INVENTOR(S): Roizman, Bernard; Chou, Joany
PATENT ASSIGNEE(S): Arch Development Corp., USA
SOURCE: PCT Int. Appl., 95 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9319591	A1	19931014	WO 1993-US1801	19930226
W: AT, AU, BB, BG, BR, CA, CH, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SK, UA				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, SN, TD, TG				
AU 9337818	A1	19931108	AU 1993-37818	19930226
AU 682463	B2	19971009		

JP 07507997 T2 19950907 JP 1993-517439 19930226
EP 675961 A1 19951011 EP 1993-907093 19930226
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
US 6172047 B1 20010109 US 1995-483533 19950607
US 6340673 B1 20020122 US 1999-283471 19990401
PRIORITY APPLN. INFO.: US 1992-861233 A 19920331
WO 1993-US1801 A 19930226
US 1995-419853 B3 19950411
US 1995-483533 A1 19950607

AB Programmed cell death in neurons is prevented or treated by gene therapy using a nonpathogenic vector contg. the .gamma.134.5 gene of herpes simplex virus type 1 (HSV-1), or by treatment with the expression product of this gene, protein ICP34.5, or its functional equivs. The function of .gamma.134.5 is to protect infected nerve cells from shutoff of protein synthesis and consequent programmed cell death, thereby promoting neuronal survival and virus replication and spreading; .gamma.134.5 and ICP34.5 may thus extend the lifetime of nonregenerating neurons in neurodegenerative diseases. The .gamma.134.5 gene and its product also protect neurons and other cells from environmental stresses which may lead to apoptosis, e.g. UV and NGF deprivation. Candidate drugs for extending the viability of cells are screened in neuroblastoma cells contg. or lacking the .gamma.134.5 gene by applying such stresses and subsequently measuring the cells' viability. The vector may be HSV-1 or HSV-2 altered by deletion of the **ICP4**, .alpha.4, or .alpha.0 gene to render it nonpathogenic, an altered retrovirus, vaccinia virus, picornavirus, coronavirus, bunyavirus, togavirus, or rhabdovirus, or a multipotent neural cell line. Thus, a mouse cerebellar progenitor cell line was transduced with replication-incompetent retroviral vector BAG contg. gene .gamma.134.5, then cocultured with a primary culture of dissocd. neonatal mouse cerebellum cells which were finally injected into newborn mice to prevent neuronal degeneration.

L11 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:249229 CAPLUS
DOCUMENT NUMBER: 118:249229
TITLE: Varicella-zoster virus open reading frame **10** **protein**, the herpes simplex virus VP16 homolog, transactivates herpesvirus immediate-early gene promoters
AUTHOR(S): Moriuchi, Hiroyuki; Moriuchi, Masako; Straus, Stephen E.; Cohen, Jeffrey I.
CORPORATE SOURCE: Lab. Clin. Invest., Natl. Inst. of Allergy and Infect. Dis., Bethesda, MD, 20892, USA
SOURCE: Journal of Virology (1993), 67(5), 2739-46
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The varicella-zoster virus (VZV) open reading frame 10 (ORF10) protein is the homolog of the herpes simplex virus type 1 (HSV-1) protein VP16. These are two virion tegument proteins that have extensive amino acid sequence identity in their amino-terminal and middle domains. ORF10, however, lacks the acidic carboxy terminus which is crit. for transactivation by VP16. Earlier studies showed that VZV ORF10 does not form a tertiary complex with the TAATGARAT regulatory element (where R is

a purine) with which HSV-1 VP16 interacts, suggesting that ORF10 may not have transactivating ability. Using transient-expression assays, it is shown that VZV ORF10 is able to transactivate VZV immediate-early (IE) gene (ORF62) and HSV-1 IE gene (**ICP4** and ICP10) promoters. Furthermore, cell lines stably expressing ORF10 complement the HSV-1 mutant in1814, which lacks the transactivating function of VP16, and enhance the de novo synthesis of infectious virus following transfection of HSV-1 virion DNA. These results indicate that ORF10, like its HSV-1 homolog VP16, is a transactivating protein despite the absence of sequences similar to the VP16 carboxy-terminal domain. The transactivating function of the VZV ORF10 tegument protein may be crit. for efficient initiation of viral infection.

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L9 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:495336 CAPLUS
DOCUMENT NUMBER: 125:140278
TITLE: Inhibition of herpes simplex virus type 1 immediate-early gene expression by alpha interferon is not VP16 specific
AUTHOR(S): Nicholl, Mary Jane; Preston, Chris M.
CORPORATE SOURCE: Medical Research Council Virology Unit, Glasgow, G11 5JR, UK
SOURCE: Journal of Virology (1996), 70(9), 6336-6339
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Pretreatment of tissue culture cells with alpha interferon (IFN-.alpha.) inhibits the transcription of herpes simplex virus type 1 (HSV-1) immediate-early (IE) genes, an effect which has been attributed to reduced **transactivation** of IE promoters by the virion protein VP16. Our previous demonstration that IFN-.alpha. inhibited the replication of the HSV-1 mutant in1814, which has a mutated VP16 unable to activate IE transcription, appeared to be incompatible with IFN-.alpha. having an effect on VP16 action (D. R. S. Jamieson, et al., 1995). To investigate this observation further, cells were infected with a deriv. of cell in1814 contg. the lacZ gene controlled by the human cytomegalovirus IE promoter. The accumulation of HSV-1 IE RNA species was inhibited by IFN-.alpha. in these cells to the same extent as in cells infected with a virus rescued at the VP16 locus, and prodn. of lacZ-specific RNA was also reduced, demonstrating that IFN-.alpha. can inhibit expression from a heterologous promoter that is not responsive to VP16. To provide a means of investigating the activity of VP16 on IE promoters not located in the HSV-1 genome, cell lines contg. the neomycin phosphotransferase gene controlled by the HSV-1 IE ICP0 promoter were constructed. Activation of the IE promoter by VP16 was not inhibited when the ICP0 promoter was resident in the cell, demonstrating that VP16 function was unaffected by pretreatment of cells with IFN-.alpha.. The results suggest that

IFN-.alpha. prevents the onset of IE transcription from the HSV-1 genome through a general mechanism rather than by having an effect specific to HSV-1 IE promoters.

L9 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:977092 CAPLUS
DOCUMENT NUMBER: 124:78443
TITLE: Amino acid substitutions in the herpes simplex virus transactivator VP16 uncouple direct protein-protein interaction and DNA binding from complex assembly and **transactivation**
AUTHOR(S): Shaw, Peter; Knez, Jozo; Capone, John P.
CORPORATE SOURCE: Dep. Biochem., McMaster Univ., Hamilton, ON, L8N 3Z5, Can.
SOURCE: Journal of Biological Chemistry (1995), 270(48), 29030-7
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The herpes simplex virus transactivator VP16 directs the assembly of a multicomponent protein-DNA complex that requires the participation of two cellular factors, the POU homeodomain protein Oct-1, which binds independently to response elements, and VCAF-1 (VP16 complex assembly factor; also called HCF, C1), a factor that binds directly to VP16. A no. of distinct properties of VP16 have been implicated in the assembly of the VP16-induced complex (VIC). These include its independent assocn. with VCAF-1 and, under appropriate conditions, its ability to bind to DNA or to DNA-bound Oct-1 in the absence of VCAF-1. In order to probe the requirements of these individual interactions in the functional assembly of VIC, we mutated selected charged amino acids in two subdomains of VP16 previously shown to be important in protein-DNA complex formation: Purified VP16 proteins were analyzed for their ability to direct protein-DNA complex formation and to interact directly with VCAF-1. Several classes of mutants that were differentially compromised in VCAF-1 interaction, direct DNA binding, and/or assocn. with DNA-bound Oct-1 were obtained. Interestingly, all of the derivs. were still capable of generating the VIC complex in vitro and activating transcription in vivo. Our findings indicate that the cooperative assembly of functional VP16-contg. complexes can occur by pathways that do not necessarily require the prior interaction of VP16 with VCAF-1 or the ability of VP16 to bind directly to DNA or assoc. with DNA-bound Oct-1.

L9 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:937803 CAPLUS
DOCUMENT NUMBER: 124:4604
TITLE: VP16 interacts via its activation domain with VP22, a tegument protein of herpes simplex virus, and is relocated to a novel macromolecular assembly in coexpressing cells
AUTHOR(S): Elliott, Gillian; Mouzakitis, Gerasimos; O'Hare, Peter
CORPORATE SOURCE: Marie Curie Res. Inst., Chart, Oxted, Surrey, RH8 0TL, UK
SOURCE: Journal of Virology (1995), 69(12), 7932-41

CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In addn. to its function as a powerful transactivator of viral immediate-early transcription, VP16 is an essential component of the herpes simplex virus (HSV) virion. As such, VP16 is introduced into cells, to effect its function in **transactivation**, as part of the virus tegument. Here we examine the potential for VP16 protein-protein interactions specific to virus-infected cells and show that VP16 copurifies in a highly enriched fraction with a single major polypeptide which we identify as the virus-encoded structural protein VP22. We further show that in vitro-translated VP22 binds specifically to purified VP16. The activation domain of VP16 was required and largely sufficient for this binding. Mutations within this domain, which disrupt its **transactivation** function, also affected VP22 binding. Furthermore, we show that while VP16 and VP22 showed distinct patterns of compartmentalization in vivo, coexpression of both proteins resulted in a profound reorganization from their normal locations to a novel macromol. assembly. The colocalization was also dependent on the activation domain of VP16 but required addnl. determinants within the N terminus. These results are discussed in the context of VP16 regulation of transcription both early in infection during delivery of tegument proteins and at late times during virus assembly.

L9 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:937771 CAPLUS
DOCUMENT NUMBER: 124:2463
TITLE: The phenotype in vitro and in infected cells of herpes simplex virus 1 .alpha. trans-inducing factor (VP16) carrying temperature-sensitive mutations introduced by substitution of cysteines
AUTHOR(S): Poon, Alice P. W.; Roizman, Bernard
CORPORATE SOURCE: Marjorie B. Kovler Viral Oncol. Lab., Univ. Chicago, Chicago, IL, 60637, USA
SOURCE: Journal of Virology (1995), 69(12), 7658-67
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB .alpha. Trans-inducing factor (.alpha.TIF, VP16, **Vmw65**) is an essential structural protein of herpes simplex virus, being required for virion assembly. The protein also forms complexes with host proteins and a response element and transactivates the .alpha. genes which carry this element. The protein contains an acidic carboxyl terminus required for **transactivation** and a much larger amino-terminal domain required for promoter recognition. We report the first set of temp.-sensitive (ts) mutations deliberately introduced into the protein by substitution of the cysteine codons with those specifying glycine at positions 78, 102, and 176, either singly or in combinations. We report the following results. (I) all mutated proteins synthesized in vitro formed complexes with the DNA response element at room temp. However, the mutant with the triple substitution and two mutants with substitutions in two of the three cysteines exhibited a ts phenotype at 33 and 37.degree.C, and one exhibited a ts phenotype only at 37.degree.C. (Ii) replacement of wild-type .alpha.TIF with genes carrying substitutions in any two cysteines conferred a ts phenotype for replication at 39.5.degree..

Shift-down expts. indicated that the 104- to 105-fold redn. in virus yield at the nonpermissive temp. was due to the disfunction of .alpha.TIF late in infection, presumably in virion maturation. (Iii) the .alpha.TIF expressed in cells infected with mutant viruses exhibited the same ts phenotype in protein-DNA complex formation as those expressed in vitro from mutated plasmids. Although the virus carrying the .alpha.TIF substitutions at Cys-102 and Cys-176 failed to induce a reporter gene linked to the .alpha.4 promoter at 39.5.degree.C, it replicated as well as the parent virus in cells maintained for the first 10 h of infection at 39.5.degree.C. We conclude the following. (I) formation of DNA-protein complexes contg. .alpha.TIF is a poor prognosticator of .alpha.TIF function. (Ii) the data presented here and in the literature strongly support the hypothesis that the secondary structure of the .alpha.TIF is very sensitive to deletions or insertions which probably affect the interaction of .alpha.TIF with both viral proteins in the virion and cellular proteins during infection. As a consequence, deletion-insertion mutagenesis may not shed useful information on the role of transactivating function of .alpha.TIF in infection. (Iii) since cysteines may play a role in stabilizing the secondary structure of proteins, substitutions of cysteines may be a powerful technique for site-specific construction of ts mutants is essential viral proteins.

L9 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:550381 CAPLUS
DOCUMENT NUMBER: 121:150381
TITLE: Protein and DNA elements involved in **transactivation** of the promoter of the bovine herpesvirus (BHV) 1 IE-1 transcription unit by the BHV .alpha. gene trans-inducing factor
AUTHOR(S): Misra, Vikram; Bratanich, Ana C.; Carpenter, Dale; O'Hare, Peter
CORPORATE SOURCE: Western Coll. Veterinary Med., Univ. Saskatchewan, Saskatoon, SK, S7N 0W0, Can.
SOURCE: Journal of Virology (1994), 68(8), 4898-909
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In herpes simplex virus (HSV)-infected cells, the transcription of immediate-early (.alpha.) genes is regulated by a virion component, the .alpha. gene trans-inducing factor (.alpha.TIF). This protein forms a complex with cellular factors and TAATGARAT motifs present in one or more copies in the promoters of all .alpha. genes. The authors have characterized the bovine herpesvirus 1 (BHV-1) homolog of this protein. Like its HSV counterpart, the BHV .alpha.TIF was synthesized in the later stages of infection and could be demonstrated to be a component of purified virions. In transient expression assays, BHV .alpha.TIF was a strong transactivator and stimulated the activity of IE-1, the major BHV-1 .alpha. gene promoter, with an efficiency comparable to that of HSV .alpha.TIF. This stimulation was largely dependent on a TAATGAGCT sequence present in a single copy in IE-1, and BHV .alpha.TIF, in conjunction with cellular factors, formed a complex with oligonucleotides contg. this sequence. Despite these similarities between the two .alpha.TIFs, the authors' preliminary observations suggest that the proteins may activate transcription by different mechanisms. Although BHV

.alpha.TIF strongly transactivated IE-1, it differed from its HSV counterpart in that the carboxyl terminus of BHV .alpha.TIF, when fused to the DNA-binding domain of GALA, was a relatively poor stimulator of a promoter contg. GAL4-binding sites. Also unlike HSV .alpha.TIF, removal of the carboxyl terminus of BZHV .alpha.TiF reduced but did not eliminate the ability of the protein to transactivate IE-1. These results are discussed in view of the structural similarities and differences among the .alpha.TIFs of alphaherpes-viruses.

L9 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:98288 CAPLUS
DOCUMENT NUMBER: 120:98288
TITLE: **Transactivation** by the herpes simplex virion component, VP16: cis- and trans-acting requirements
AUTHOR(S): ap Rhys, Collette Marie Jose
CORPORATE SOURCE: John Hopkins Univ., Baltimore, MD, USA
SOURCE: (1992) 218 pp. Avail.: Univ. Microfilms Int., Order No. DA9229393
From: Diss. Abstr. Int. B 1992, 53(5), 2160
DOCUMENT TYPE: Dissertation
LANGUAGE: English
AB Unavailable

L9 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:667857 CAPLUS
DOCUMENT NUMBER: 119:267857
TITLE: The octamer binding protein Oct-2 inhibits **transactivation** of the herpes simplex virus immediate-early genes by the virion protein **Vmw65**
AUTHOR(S): Lillycrop, K. A.; Estridge, J. K.; Latchman, D. S.
CORPORATE SOURCE: Med. Sch., Univ. Coll. London, London, W1P 6DB, UK
SOURCE: Virology (1993), 196(2), 888-91
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Transactivation** by a complex of the cellular transcription factor Oct-1 and the virion protein **Vmw65** is necessary for the high-level activity of the HSV immediate-early promoters during lytic infection. The authors show that the **transactivation** can be inhibited by two forms of the Oct-2 transcription factor which are expressed at high levels in neuronal cells as well as by the isolated DNA binding, POU domain of Oct-2. The inhibition of Oct-1-**Vmw65** DNA binding by these neuronal forms of Oct-2 is likely to play a crit. role in the nonpermissivity of neuronal cells for the HSV lytic cycle and therefore in the establishment of latent infections.

L9 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:600866 CAPLUS
DOCUMENT NUMBER: 119:200866
TITLE: **Transactivation** by the herpes simplex virus virion protein **Vmw65** and viral permissivity in a neuronal cell line with reduced levels of the cellular transcription factor Oct-1
AUTHOR(S): Howard, M. Keith; Mailhos, Carolina; Dent, Carolyn L.;
Latchman, D. S.

CORPORATE SOURCE: Dep. Biochem., Univ. Coll., London, W1P 6DB, UK
SOURCE: Experimental Cell Research (1993), 207(1), 194-6
CODEN: ECREAL; ISSN: 0014-4827
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Transactivation** of the herpes simplex virus (HSV) immediate-early (IE) genes is dependent upon the formation of a complex between the viral protein **Vmw65** and the cellular **transactivation** factor Oct-1. Differentiation of the proliferating ND7 neuronal cell to a nondividing phenotype results in a large fall in the amt. of Oct-1 to a level characteristic of nondividing neuronal cells but does not dramatically affect the level of IE gene expression following infection or the ability of **Vmw65** to transactivate the IE promoter in transfection expts. This suggests that the low levels of Oct-1 in nonproliferating neuronal cells do not play a key role in the failure of IE gene expression following initial infection of these cells, which is an essential step in the est. of latent infections with HSV.

L9 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:532664 CAPLUS
DOCUMENT NUMBER: 119:132664
TITLE: Cross-hybridization of equid herpesvirus-2 (EHV-2) and herpes simplex virus-1 (HSV-1) genes to equid herpesvirus-1 (EHV-1)
AUTHOR(S): Purewal, A. S.; Smallwood, A. V.; Allsopp, R.; Welch, H. M.; Edington, N.
CORPORATE SOURCE: Dep. Vet. Pathol., R. Vet. Coll., London, UK
SOURCE: Veterinary Microbiology (1993), 35(1-2), 1-10
CODEN: VMICDQ; ISSN: 0378-1135
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In contrast to previous findings, the Ab4 isolate of equid herpesvirus-1 (EHV-1) was shown to share homol. with the G9 isolate of equid herpesvirus-2 (EHV-2). Using Southern blotting and stringent hybridization conditions, a significant proportion of this cross-hybridization was identified by the immediated-early gene-3 (IE-3) probe from herpes simplex virus-1 (HSV-1). The HSV-1 UL48 gene probe (encoding the IE gene transactivating protein **VmW65**, which is also known as .alpha.-TIF or VP16) was used to identify and isolate its counterpart in EHV-1. The relevance of shared homol. to **transactivation** is being investigated.

L9 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:189097 CAPLUS
DOCUMENT NUMBER: 116:189097
TITLE: Identification of dominant-negative mutants of the herpes simplex virus type 1 immediate-early protein ICP0
AUTHOR(S): Weber, Peter C.; Wigdahl, Brian
CORPORATE SOURCE: Coll. Med., Pennsylvania State Univ., Hershey, PA, 17033, USA
SOURCE: Journal of Virology (1992), 66(4), 2261-7
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB ICP0 is a 110,000-mol.-wt. immediate-early protein of herpes simplex virus type 1 (HSV-1) which is encoded by three exons. It functions as a

promiscuous transactivator of a variety of different HSV-1 and non-HSV-1 promoters in transient expression assays. Anal. of mutations which truncated the carboxy-terminal end of this 775-amino-acid (aa) protein demonstrated that a polypeptide which contained only aa 1 to 553 still possessed significant **transactivation** potential. Addnl. carboxy-terminal truncations which sequentially removed aa 245 to 553 and thus the remainder of the third exon resulted in the eventual loss of **transactivation** capability in these mutants. However, further anal. of these truncated derivs. demonstrated that they behaved as dominant-neg. mutants to the wild-type polypeptide. Moreover, one of the mutants was found to act as a promiscuous repressor, in that it could dramatically inhibit a variety of HSV-1 promoters, non-HSV-1 promoters, and heterologous transactivator proteins in transient expression assays, despite having lost almost the entire third exon. These results indicate that a domain encoded by the first two exons probably interacts with, and can effectively titrate, the unknown cellular factor(s) through which ICP0 mediates **transactivation**.

L9 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:179617 CAPLUS
DOCUMENT NUMBER: 114:179617
TITLE: High efficiency transient expression of eukaryotic genes: use of an HSV-1 immediate early promoter (ICP4)
AUTHOR(S): Liu, Xuan; Giza, Christopher C.; Vrana, Kent E.
CORPORATE SOURCE: Health Sci. Cent., West Virginia Univ., Morgantown, WV, 26506, USA
SOURCE: BioTechniques (1990), 9(2), 168-70, 172-3
CODEN: BTNQDO; ISSN: 0736-6205
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The transcriptional efficiencies of a no. of eukaryotic promoters were compared following DNA-mediated transfection into cultured rat hepatoma cells. The highest levels of expression for the bacterial chloramphenicol acetyltransferase (CAT) reporter gene are obsd. with a herpes simplex virus type 1 (HSV-1) immediate early promoter when co-transfected with an expression construct bearing the gene for the HSV-1 transcriptional activator protein VP16. This **transactivation** phenomenon is specific for the HSV-1 immediate early promoter and increases the expression of the reporter gene 7-fold. Expression from the ICP4 promoter is 2.5-fold greater than the other promoters tested. In addn., expression from the ICP4 promoter can be induced, at varying times followed transfection, by infecting the cells with HSV-1 viral particles. Two plasmids have been constructed which contain the HSV-1 ICP4 promoter adjacent to a multiple cloning site. One of the plasmids also contains SV40 splicing and polyadenylation signals.

L9 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1990:493403 CAPLUS
DOCUMENT NUMBER: 113:93403
TITLE: Direct and selective binding of an acidic transcriptional activation domain to the TATA-box factor TFIID
AUTHOR(S): Stringer, Keith F.; Ingles, C. James; Greenblatt, Jack
CORPORATE SOURCE: Banting and Best Dep. Med. Res., Univ. Toronto,

Toronto, ON, M5G 1L6, Can.
SOURCE: Nature (London, United Kingdom) (1990), 345(6278), 783-6
CODEN: NATUAS; ISSN: 0028-0836
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Herpes simplex virion protein VP16 activation domain binding by human and yeast TATA box factors was highly selective and useful for purifn. of TFIID by affinity chromatog.

L9 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:186934 CAPLUS
DOCUMENT NUMBER: 110:186934
TITLE: Separation of requirements for protein-DNA complex assembly from those for functional activity in the herpes simplex virus regulatory protein **Vmw65**
AUTHOR(S): Greaves, R.; O'Hare, P.
CORPORATE SOURCE: Marie Curie Res. Inst., Oxted/Surrey, RH8 OTL, UK
SOURCE: J. Virol. (1989), 63(4), 1641-50
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A transient expression system was developed which results in efficient synthesis of the regulatory protein **Vmw65** of herpes simplex virus type 1 in eucaryotic cells. The gene for **Vmw65** was linked to the cytomegalovirus immediate-early (IE) promoter-enhancer region in a plasmid contg. the simian virus 40 origin of replication. When transfected into COS cells, **Vmw65** was expressed from this vector in 25 to 50% of the cells, with total levels of the protein approaching 20% of those obsd. in infected cells. **Vmw65** expressed in this system is functional for specific DNA-binding complex formation with the host cell octamer-binding protein TRF and for **transactivation** of IE gene expression. Therefore a series of carboxy-terminal truncated forms of **Vmw65** was produced to examine the structural requirements of the protein for these activities. Deletion of the acidic carboxy-terminal 56 amino acids had no effect on DNA-binding complex formation but completely abolished the ability to transactivate. Amino acids between residues 434 and 453, a region which exhibits a high neg. charge, were crit. for IE **transactivation**. In contrast, the requirements for complex formation are located entirely within the N-terminal 403 amino acids, and the results indicate a requirement for this activity for residues between 316 and 403. Together with previous work, the results presented indicate that recruitment of TRF into a specific DNA-binding complex on IE consensus signals is required but not sufficient for functional IE **transactivation** by **Vmw65**.

L9 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:52087 CAPLUS
DOCUMENT NUMBER: 110:52087
TITLE: Expression of a truncated viral trans-activator selectively impedes lytic infection by its cognate virus
AUTHOR(S): Friedman, Alan D.; Triezenberg, Steven J.; McKnight, Steven L.
CORPORATE SOURCE: Dep. Embryol., Carnegie Inst. Washington, Baltimore, MD, 21210, USA
SOURCE: Nature (London) (1988), 335(6189), 452-4
CODEN: NATUAS; ISSN: 0028-0836
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A virion protein of herpes simplex virus type 1 (HSV-1) specifically and potently activates transcription of the viral immediate early genes. Appropriate function of this protein, termed VP16, depends on an acidic transcriptional activation domain located within the 78 carboxyl-terminal amino acids of the protein. Mutated forms of the protein lacking this acidic domain lose the ability to activate transcription, and can dominantly interfere with the trans-activation function of native VP16. Stably transformed mouse L cells that constitutively express a form of VP16 lacking its acidic activating domain were prepd. These cells are selectively impaired in their capacity to support the lytic infectious cycle of HSV-1, and this impairment results from their inability to support immediate early transcription.

L9 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1988:181445 CAPLUS
DOCUMENT NUMBER: 108:181445
TITLE: Herpes simplex virus regulatory elements and the immunoglobulin octamer domain bind a common factor and are both targets for virion **transactivation**
AUTHOR(S): O'Hare, P.; Goding, C. R.
CORPORATE SOURCE: Marie Curie Res. Inst., Surrey, UK
SOURCE: Cell (Cambridge, Mass.) (1988), 52(3), 435-45
CODEN: CELLB5; ISSN: 0092-8674
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Functional upstream activator sequences (TAATGARAT motifs) of herpes simplex virus immediate-early genes were identified and shown both to bind a factor (TRF) present in uninfected HeLa cells and to confer inducibility by the virus regulatory protein, **Vmw65**, on a normally nonresponsive promoter. Point mutation analyses demonstrated binding specificity and correlated binding with **Vmw65** induction. Furthermore, the octamer domains of the adenovirus DNA replication origin, the histone H2B, and the Ig light chain genes bound and competed for TRF. The Ig octamer also conferred **Vmw65** inducibility on the TK promoter. In addn., a modified form of TRF was specifically detected in infected cells. Apparently, TRF is similar or identical to the previously described octamer-binding protein and is likely to be the target for coordinate induction of immediate-early gene expression by **Vmw65**.

L9 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:124043 CAPLUS
DOCUMENT NUMBER: 104:124043
TITLE: Analysis of DNA sequences which regulate the transcription of herpes simplex virus immediate early gene 3: DNA sequences required for enhancer-like activity and response to trans-activation by a virion polypeptide
AUTHOR(S): Bzik, David J.; Preston, Chris M.
CORPORATE SOURCE: Virol. Unit, Med. Res. Counc., Glasgow, G11 5JR, UK
SOURCE: Nucleic Acids Res. (1986), 14(2), 929-43
CODEN: NARHAD; ISSN: 0305-1048
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The far upstream region of herpes simplex virus (HSV) immediate early (IE)

gene 3 has previously been shown to increase gene expression in an enhancer-like manner, and to contain sequences which respond to stimulation of transcription by a virion polypeptide, **Vmw65**. To analyze the specific DNA sequences which mediate these functions, sequential deletions from each end of the far upstream region were made. The effects of the deletions on transcription in the absence or presence of the **Vmw65** were measured by use of a transient expression assay. The enhancer-like activity was due to 3 separable elements, whereas 2 addnl. DNA regions were involved in the response to **Vmw65**. One of the responding elements corresponded to an AT-rich consensus (TAATGARATTC, where R=purine) present in all IE gene far upstream regions, and the other was a GA-rich sequence also present in IE genes 2 and 4/5. The TAATGARATTC element could mediate responsiveness to **Vmw65** but it was fully active only in the presence of the GA-rich element. The GA-rich element was unable to confer a strong response alone but could activate an otherwise nonfunctional homolog of TAATGARATTC.

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L8 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:920110 CAPLUS
DOCUMENT NUMBER: 136:165922
TITLE: Infection of murine keratinocytes with herpes simplex virus type 1 induces the expression of interleukin-10, but not interleukin-1.alpha. or tumor necrosis factor-.alpha.
AUTHOR(S): Zak-Prelich, Malgorzata; Halliday, Katrina E.; Walker, Craig; Yates, Catherine M.; Norval, Mary; Mckenzie, Roddie C.
CORPORATE SOURCE: Department of Dermatology, Medical University of Lodz, Lodz, Pol.
SOURCE: Immunology (2001), 104(4), 468-475
CODEN: IMMUAM; ISSN: 0019-2805
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Herpes simplex virus (HSV) is known to possess several mechanisms whereby it can evade the normal host immune defenses. Here, the expression of the immunosuppressive cytokine, interleukin (IL)-10, was monitored following infection of a murine keratinocyte cell line (PAM-212) and compared with the expression of 2 proinflammatory cytokines: IL-1.alpha. and tumor necrosis factor (TNF)-.alpha.. The PAM-212 cells were infected at a multiplicity of 0.5 with a clin. isolate of HSV type 1, and the mRNA of the 3 cytokines was assessed by semiquant. reverse transcription-polymerase chain reaction (RT-PCR) over the following 24 h. By 12 h postinfection the amt. of IL-10 mRNA had increased to 5-fold greater than that found in uninfected cells, and this elevated level was maintained until at least 24 h postinfection. In contrast, IL-1.alpha. and TNF-.alpha. mRNAs were not up-regulated by the HSV infection. Immunostaining with an IL-10 monoclonal antibody (mAb) revealed that cytoplasmic IL-**10 protein** had increased by 6-12 h

postinfection. This quantity was further increased at 24 h postinfection, when the viral cytopathic effect was apparent. Viral replication was necessary, but not sufficient on its own, for IL-10 induction. Expts. with HSV mutants lacking functional transactivating factors suggested that the viral transactivating proteins ICP-0 and VP-16 may be necessary for HSV-induced IL-10 expression. Thus, the up-regulation in the expression of IL-10 mRNA and protein induced by HSV early in the infection of keratinocytes represents a specific response and may be part of the viral strategy to avoid local immune defense mechanisms in the skin.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1992:468360 CAPLUS
DOCUMENT NUMBER: 117:68360
TITLE: Non-pathogenic herpes simplex viruses for use in vaccines
INVENTOR(S): Roizman, Bernard
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9204050	A1	19920319	WO 1991-US6532	19910910
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
US 5328688	A	19940712	US 1990-579834	19900910
CA 2072627	AA	19920311	CA 1991-2072627	19910910
AU 9187418	A1	19920330	AU 1991-87418	19910910
AU 658838	B2	19950504		
EP 500917	A1	19920902	EP 1991-918320	19910910
EP 500917	B1	19970502		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 05503017	T2	19930527	JP 1991-516968	19910910
AT 152355	E	19970515	AT 1991-918320	19910910
ES 2102409	T3	19970801	ES 1991-918320	19910910
US 6120773	A	20000919	US 1994-272772	19940708

PRIORITY APPLN. INFO.: US 1990-579834 A 19900910
WO 1991-US6532 A 19910910

AB Herpes simplex virus carrying alleles of the gene for ICP34.5 that encode an inactive form of the protein are avirulent and suitable for use in live vaccines. The gene is found in the long terminal repeat of the viral genome and is essential for pathogenicity but not for propagation in cell culture. A series of derivs. of herpes simplex virus 1 in which the gene for this protein (.gamma.134.5) was disrupted by insertion, deletion, or introduction of stop codons into possible reading frames were prepd. These derivs. showed no significant changes in plaque morphol. in cell culture. The pfu/LD50 for these derivs. in mouse intracranial inoculation increased from 420 for the wild type to >106. A deriv. with a monoclonal

antibody epitope at the N-terminus had a pfu/LD50 of 4200.

L8 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:103281 CAPLUS
DOCUMENT NUMBER: 104:103281
TITLE: Multistep transformation by defined fragments of herpes simplex virus type 2 DNA: oncogenic region and its gene product
AUTHOR(S): Hayashi, Yoshinobu; Iwasaka, Tsuyoshi; Smith, Cynthia C.; Aurelian, Laure; Lewis, George K.; Ts'o, Paul O. P.
CORPORATE SOURCE: Div. Biophys., Johns Hopkins Med. Inst., Baltimore, MD, 21205, USA
SOURCE: Proc. Natl. Acad. Sci. U. S. A. (1985), 82(24), 8493-7
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Diploid Syrian hamster embryo cells transfected with BglII C fragment of herpes simplex virus type 2 DNA acquired a neoplastic phenotype. Cultures transfected with its left-hand 64% subclone EcoRI/HindIII fragment AE (0.419-0.525 map unit) grew into established but nontumorigenic lines. Transfection of EcoRI/HindIII AE-immortalized cells with a 4.4-kilobase SacI/BamHI subfragment within BamHI E (0.554-0.584 map unit; overlaps the right-hand 16% of BglII C) converted them to tumorigenicity. The 4.4-kilobase subfragment encodes a 144-kilodalton (kDa) protein immunol. and structurally similar to an infected cell protein designated ICP 10. DNA extd. from cells transformed with the 4.4-kilobase subfragment exhibited discrete hybridizing bands homologous to BamHI E fragment. Monoclonal antibody to ICP 10 pptd. a 144-kDa protein from the transformed cells and stained them in immunofluorescence. A tumor deriv. established with the transformed cells did not stain with this antibody, but .apprx.25% of the cells stained with a monoclonal antibody to c-myc protooncogene products.

L8 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:573797 CAPLUS
DOCUMENT NUMBER: 99:173797
TITLE: Expression and cellular compartmentalization of a herpes simplex virus type 2 protein (ICP 10) in productively infected and cervical tumor cells
AUTHOR(S): Aurelian, L.; Smith, C. C.; Klacsman, K. T.; Gupta, P. K.; Frost, J. K.
CORPORATE SOURCE: Dep. Biochem. Biophys., Johns Hopkins Med. Inst., Baltimore, MD, 21205, USA
SOURCE: Cancer Invest. (1983), 1(4), 301-13
CODEN: CINVD7; ISSN: 0735-7907
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Antiserum to ICP 10, a herpes simplex virus type 2 (HSV-2) protein that is expressed in cells neoplastically transformed by viral DNA sequences within the Bgl II/Hpa 1 CD fragment, specifically ppts. the ICP **10 protein** from HSV-2 infected cells and stains cells infected with HSV-2 for 4-16 h by indirect immunofluorescence. At 4 h post infection (p.i.), the staining is primarily perinuclear, while at 16 h p.i., it is

cytoplasmic and intranuclear. Compartmentalization studies indicate that the [35S]L-methionine labeled ICP 10 is detectable in both the cytoplasmic and nuclear fractions early and late in infection. However, in its phosphorylated form, ICP 10 is undetectable in the nuclear fraction late in the viral reproductive cycle. Anti-ICP 10 serum stains a high (75-83%) proportion of cervical tissue with pathol. findings of dysplasia or carcinoma, as well as atypical exfoliated cells from these patients. Cervical tumor tissue from 4 of 12 patients also stains with antiserum to another purified viral protein complex designated ICP 12114. In the majority of atypical cells with mild or moderate changes, ICP 10 localizes in the cytoplasm, while the majority of atypical cells with severe changes also display nuclear staining with anti-ICP 10 serum. While exfoliated atypical cells from 60% of patients with dysplasia are pos. for ICP 10, those from only 50% of these patients stain also with anti-ICP 12114 serum and this staining is strictly cytoplasmic. Atypical cells from 3 patients in these series stain with the anti-HSV-2 serum but are neg. for both ICP 10 and ICP 12114. Exfoliated atypical cells from patients with carcinoma in situ or invasive cancer stain equally well with all 3 antisera.

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